Case Report

Hemolytic Uremic Syndrome- a rare complication of Viper envenomation

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Abstract:
The triad of acute renal failure, thrombocytopenia and haemolytic anaemia with schistocytes (fragmented erythrocytes) comprise the Haemolytic Uraemic Syndrome (HUS).Its pathogenesis involves an activation/lesion of microvascular endothelial cells, mainly in the renal vasculature, secondary to bacterial toxins, drugs, or autoantibodies. Less frequently, renal microthrombi are due to an acquired or a constitutional deficiency in ADAMTS-13, the protease cleaving Von Wilebrand factor H. In adults, several HUS are encountered in the course of chronic nephropathies: nephroangiosclerosis, chronic glomerulonephritis, post irradiation nephropathy, scleroderma, disseminated lupus erythematosus, antiphospholipid syndrome, and toxins. Coagulopathy is the commonest important, systemic clinical syndrome caused by snake envenoming in the world, and venom-induced consumption coagulopathy (VICC) is the most clinically important coagulopathy but rare , because it can be complicated by serious and life-threatening haemorrhage .We report a rare case of a farmer developing HUS after 48 hours following Viper bite.

Key Words: Viper bite, consumptive coagulopathy, thrombotic microangiopathy

Introduction:

Viper bite is known to cause coagulopathy, acute renal failure, acute respiratory distress syndrome, and gangrene of the distal limb[1.] Venom-induced consumptive coagulopathy (VICC) is the commonest coagulopathy that occurs following snake envenomation which is characterised by prolonged clotting time. In a small proportion of patients with VICC, microangiopathy is also seen[2.] The combination of acute renal failure, thrombocytopenia and microangiopathic haemolytic anaemia characterises thrombotic microangiopathy and in snake envenoming it usually occurs in association with VICC. We report a rare case of a farmer developing HUS after 48 hours following Viper envenomation.

Case Report:

A 55 year old agriculturist presented to us with history of viper bite two days prior. On examination he had bite mark in left lower limb with minimal local reaction. He had features of systemic envenomation and was in respiratory distress. On investigation he had anemia, coagulopathy, thrombocytopenia and acute renal failure. He was started on polyvalent antisnake venom, mechanical ventilation and hemodialysis. His renal failure, anaemia and thrombocytopenia worsened and jaundicedeveloped. On further investigation his Creatinine Phosphokinase and Lactate Dehydrogenase levels were grossly elevated and the peripheral smear showed evidence of hemolysis in the form of schistocytes. D dimer was positive. Septic work up was negative. A diagnosis of Hemolytic Uremic Syndrome secondary to snake bite was made and was initiated on plasmapheresis. Patient received 5 sessions of plasmapheresis and he improved gradually over the next 15 days. At the time of discharge the renal functions, blood counts and coagulation profile were back to normal.

Discussion:

Viper is a moderately venomous snake found in South India. Most victims show local swelling, pain and bleeding. Haemorrhagic blisters are seen at the bite site and rarely have led to amputations[1.] However, there are several reports of coagulopathy, thrombocytopenia and acute renal failure occurring in isolation and in combination after viper envenomation[3-5.]The combination of acute renal failure, thrombocytopenia and microangiopathic haemolytic anaemia characterises thrombotic microangiopathy and in snake envenoming it usually occurs in

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association with VICC. Coagulopathy in snake envenomation depends on which point the coagulation cascade is activated by the procoagulant snake toxin. This is a major difference inVICC from where coagulation is activated by tissue factor/factor VII pathway [6.] It is difficult to predict the type of coagulopathy from the envenomation as most snake venom contains a mixture of procoagulant toxins acting at different points of the coagulation cascade. Apart frombasic clotting tests, few laboratory data are available on the coagulopathy of snake venom in clinical studies. Out of procoagulant toxins present in viper venom, Thrombin Like Enzyme(TLE) mainly causes hypofibrogenaemia[7.]Often this hypofibrogenaemia is associated with thrombocytopenia [8.] Considering our patient's thrombocytopenia and elevated D dimer levels we assume that viper venom contains predominantly TLEs as procoagulant toxin. Coagulopathy in VICC has a rapid onset and resolves within 24-48 hours if prompt treatment is done. Despite the resolution of the VICC, thrombotic microangiopathy (TMA) progresses. This suggests an involvement of a different toxin to procoagulant toxin initiating TMA [6]. In combination with renal failure this clinical picture is similar to haemolytic uremic syndrome which usually results from a toxin causing renal endothelial damage. It is possible that a toxin in venom is causing a similar endothelial damage initiating the thrombotic microangiopathy[9.]

The treatment for russel viper envenomation is supportive with antivenom. Acute renal failure is treated with dialysis and therapeutic transfusions as required. HUS has to be treated with plasmapheresis. [9]

Conclusion:

Our case highlights the need for comprehensive and serial hematological evaluation in a patient with viper bite to detect Haemolytic Uraemic Syndrome (HUS) and to initiate timely plasma exchange**.**

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